Language disorders: **A problem with auditory processing?**

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Recent studies have found associations between auditory processing deficits and language disorders such as dyslexia; but whether the former cause the latter, or simply co-occur with them, is still an open question.

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Dyslexia is commonly described as a disorder manifested by difficulties in learning to read and spell, despite adequate intelligence and conventional instruction. It is often diagnosed on the basis of a discrepancy between measures of reading ability and other cognitive skills, and is said to occur in 4–7% of children [1]. Explanations for dyslexia fall into three main categories. Perhaps the most popular current thinking among scientists ascribes dyslexia to an underlying deficit in the representation and processing of information about speech sounds, typically referred to as 'phonological processing' [1]. A deficit in phonological processing is reflected in poor performance in tasks such as reading non-words (which requires knowledge of letter-to-sound mappings), repeating back nonsense words presented auditorily, in judging whether words rhyme and breaking up words into their component sounds. In this view the core deficit is seen to be linguistic, as it applies specifically to an aspect of language processing.

Other proposed explanations of dyslexia stress underlying perceptual difficulties in vision or audition. Attempts to implicate defects in visual processing have, at least so far, been specific to dyslexia, but auditory deficits have been suggested to occur in a much wider variety of language disorders. The auditory deficit view goes back at least to the early 60s. In a groundbreaking paper, Robert Efron [2] attributed the language difficulties of patients with acquired aphasia — where a loss of language ability has been caused by a brain injury — to impairments of rapid auditory processing, as measured in a task that required judgement of the temporal order of auditory stimuli. Two recent studies [3,4] have provided evidence about the locus of the auditory deficit often associated with developmental dyslexia, though whether the association really reflects causality remains unclear.

It is important to note that the concept of 'auditory deficit' does not refer to the kinds of hearing loss associated with peripheral hearing impairment. Although it is clear that such hearing losses have a significant impact on the development of language skills, the auditory deficits discussed here are ones which imply an inability to discriminate between sounds, as opposed to simply requiring the sounds to be louder in order for them to be heard. It is also supposed that these auditory deficits reflect disordered processing at a neural site more central than the inner ear.

Efron's approach was advanced most diligently and consistently by Paula Tallal and her colleagues in studies of children with 'specific language impairment', originally known as developmental dysphasia/aphasia. Like dyslexia, specific language impairment is defined by a reasonably specific deficit in language-related abilities, in the presence of relatively intact non-linguistic cognitive abilities. Here though, the deficit appears more generally in language acquisition and use, for example, in subject–verb agreement — "The boy jump the fence" — and tense marking — "Yesterday I rush around London".

Tallal and Piercy [5] modified Efron's order-judgement task to create what is known as the repetition task. In this task, listeners first learn to differentially label, by pressing buttons, two distinct sounds, in their case 70 millisecond complex tones differing in fundamental frequency and hence perceived pitch. Then two sounds are presented sequentially, with a varying inter-stimulus interval, and the listener has to indicate which sounds were presented by pushing two buttons sequentially. Unlike temporal order judgement tasks, where the only two kinds of trial would be sound 1 followed by sound 2 and the reverse, here the two sounds can be the same, making for a total of four different trial types: 1–2, 2–1, 1–1 or 2–2. The crucial aspect of Tallal and Piercy's result was that the children exhibiting specific language impairment showed abnormal difficulty in performing the task only when inter-stimulus intervals were short (≤ 300 milliseconds), and performed near perfectly at longer inter-stimulus intervals.

These results lent support to the notion that specific language impairment is associated with a deficit for the processing of rapid auditory sequences, and that this deficit might underlie the linguistic problem. Such ideas have also played a crucial role in theorizing about the underlying causes of dyslexia, without necessarily contradicting the notion that a phonological deficit lies at the heart of dyslexia. It is easy to imagine that an impairment of auditory processing could affect the development of the ability the detect and process the dynamic acoustic patterns of

speech, leading to impaired phonological processing and thereby to problems in reading.

The two recent studies [3,4] addressed different aspects of the idea that dyslexia can be explained by an underlying sensory limitation. I shall first discuss the work of Nagarajan *et al.* [3], which has provided direct physiological evidence that an impairment quite early in the auditory pathway can be demonstrated in at least some dyslexic adults. Following that, I shall discuss the work of Witton *et al.* [4], which has provided behavioural evidence that deficits in auditory and visual processing are not independent factors associated with dyslexia, but in fact are correlated and so may arise from a common source.

Nagarajan *et al.* [3] made magnetoencephalographic (MEG) recordings from sensors placed over the left temporal area while their adult listeners performed the repetition task described above. The two sounds were 20 millisecond sinusoids of frequencies 800 Hz and 1200 Hz; the inter-stimulus intervals ranged from 100–500 milliseconds. A number of differences were found between the seven subjects with dyslexia and seven controls, but I shall focus here on the MEG peak found approximately 100 milliseconds after the stimulus, known as the 'M100 response' ('M' for 'magnetic'). Crucially, these responses were found to arise from the primary auditory cortex.

There were a number of significant differences between the MEG waveforms recorded from control and dyslexic listeners. For one thing, when the two sounds were 500 milliseconds apart, there was a greater response for the dyslexic listeners at about 200 milliseconds post-stimulus. A more directly interpretable difference was the much smaller response of dyslexic listeners to the second sound when the inter-stimulus interval was 200 milliseconds, which may indicate a less robust response to short sounds preceded quickly by another sound. These and other differences led Nagarajan *et al.* [3] to conclude that "reading disabilities are correlated with the abnormal neural representation of brief and rapidly successive sensory inputs, manifested in this study at the entry level of the cortical auditory/aural speech representational system(s)".

At least as striking to a behavioral auditory scientist such as myself is the poor performance of the dyslexic listeners. With an inter-stimulus interval of 500 milliseconds, their mean accuracy was below 80%, dropping to below 60% when the inter-stimulus interval was reduced to 100 milliseconds. So contrary to conclusions of Tallal and Piercy [5], the auditory deficit is not specific to rapidly presented stimuli (although separate testing showed that all the listeners could accurately label the tones when presented at inter-stimulus intervals greater than a second; S. Nagarajan, personal communication). Control listeners made few or no errors in this task, and from previous studies one would expect the levels of performance to be above 90% correct down to inter-stimulus intervals of less than a millisecond, at least for practiced listeners [6]. Such poor abilities might be thought to be devastating for the perception of speech, but no evidence for this is presented, nor is there any evidence in the literature that most people with dyslexia, whether children or adults, have a serious deficit in the perception of speech.

Nagarajan *et al.* [3] conclude that their results reveal differences in neuronal representation between people with dyslexia and controls, but it should be noted the dyslexic listeners were also selected on the basis of poor auditory performance. The question then naturally arises as to whether the abnormal neural processing is associated primarily with the behaviourally-measured auditory deficit, rather than with dyslexia *per se*. Nagarajan *et al.* [3] make strong claims on this score, that at least 90% of poor-reading adults show such "abnormal perceptual processing of brief, rapidly successive stimuli". This estimate is much higher than is indicated in the previous literature, which typically indicates that less than half of those with dyslexia have an auditory deficit. Of course, it may well be that this particular auditory task is more sensitive than others that have been used, or that dyslexic children with normal auditory processing abilities 'outgrow' their reading problems (although current opinion is moving to the notion that dyslexia is a brain-based deficit which persists through life).

In their recent study, Witton *et al.* [4] have addressed this issue, at least incidentally. They investigated the perception of dynamic acoustic patterns in a frequency-modulation detection task. Here the minimum detectable level of 2 Hz frequency modulation of a 500 Hz sinusoidal tone was determined for a group of 17 adults with dyslexia and 18 controls. They found a reasonable correlation between a measure of non-word reading ability and the frequency-modulation detection threshold, but only about half of the dyslexic listeners showed a significantly lower performance level than control listeners. Perhaps more importantly, a correlation was observed between abilities at detecting auditory frequency modulation and visual coherent motion, another dynamic sensory pattern. (But note that the correlation found depends heavily on the four dyslexic subjects with the highest motion detection thresholds — if they are removed, there is a non-significant correlation of approximately 0.3).

It would be hard to argue from these results that impaired visual and auditory processing universally underlies dyslexia across the affected population, given that about half of those with dyslexia have apparently normal auditory and visual processing abilities. What is more thought-provoking is the hypothesis advanced to explain the observed deficits. Drawing on earlier work by Stein [7], it

is proposed that both the auditory and visual deficits arise from abnormalities in the large cells — 'magnocells' — of the auditory and visual relay nuclei. From this point of view, visual, auditory and phonological deficits can be seen to be different expressions of the same underlying cause, rather than competing hypotheses. And, of course, abnormalities of processing in auditory brainstem nuclei would be reflected in primary auditory cortex.

Although little is understood about the relationship between the two kinds of auditory tasks described above that people with dyslexia appear to find difficult, it is interesting to note that Nagarajan *et al.* [3] stress that it is rapid auditory processing that is impaired. However, neither their stimulus configurations, nor those of Witton *et al.* [4] — occurring over hundreds of milliseconds — can be called particularly rapid. In fact, deficits over this time scale appear in keeping with the results of a recent extensive study of the perception of speech in dyslexic children by Adlard and Hazan [8]. They found difficulties for a variety of phonetic contrasts, not just those characterized by rapid acoustic patterning. Importantly for the argument about the ubiquity of auditory processing deficits in dyslexia, only 30% of this group of dyslexic children differed significantly from controls in their speech-perception abilities.

Which brings us to the 64-million-dollar question. Although dyslexia is often accompanied by an auditory processing deficit, what is much less clear is the extent to which this deficit actually causes the dyslexia. Two complementary approaches may clarify this situation. One is to assess the incidence of poor auditory processing in people who read normally. And the other is to identify and fully characterize people with dyslexia whose auditory processing abilities appear to be intact.

A variant of the second strategy — the characterization of 'pure' cases of a disorder — is exemplified in a recent study of children with specific language impairment by van der Lely *et al.* [9]. Here a subgroup of seven children with specific language impairment has been identified, whose *only* deficit appears to be in grammar. Although auditory testing of only one child was reported in that paper, his auditory processing ability was normal in two tasks previously shown to distinguish children with specific language impairment from normally-developing children. Furthermore, my group's current studies of fourteen other children with such a 'grammar-specific language impairment' shows many to have intact auditory processing abilities. As the form of the grammatical deficit does not vary with the presence or absence of auditory problems, it appears that the core deficit of these children is specifically grammatical and that the auditory processing deficits sometimes found are not the root of their language problems.

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